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DEVELOPMENT OF A CLINICAL ALGORITHM FOR MONITORING AND PROGNOSTIC ASSESSMENT IN PATIENTS WITH CKD AFTER COVID-19

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ABSTRACT

Introduction Chronic kidney disease and coronavirus disease 2019 represent a high-risk intersection, yet renal follow-up after coronavirus disease 2019 remains insufficiently standardized. We developed and internally validated a risk-stratified clinical algorithm for monitoring and prognostic assessment of patients with chronic kidney disease after coronavirus disease 2019.

Materials and methods This was a prospective cohort study of 280 non-dialysis patients with chronic kidney disease stages G2-G5: 140 post-coronavirus disease 2019 and 140 controls. At 6 to 12 months, we assessed estimated glomerular filtration rate, proteinuria, and inflammatory, metabolic, and hematologic markers. Coronavirus disease 2019 severity and the occurrence of acute kidney injury during infection were recorded. Predictors of advanced chronic kidney disease stages G4-G5 were identified by multivariate logistic regression; algorithm performance was evaluated by calibration and receiver operating characteristic analysis.

Results The decline in renal function was comparable between groups. In post-coronavirus disease 2019 patients, persistent proteinuria, elevated serum phosphate, and low hemoglobin independently predicted advanced chronic kidney disease (odds ratio 5.0, 1.8, 1.4, respectively ($p<0.005$). The model showed good internal performance (area under the curve approximately 0.88).

Conclusions Coronavirus disease 2019 appears to amplify renal vulnerability. A structured, risk-based algorithm may improve early detection of renal deterioration and support personalized management.

Keywords Chronic kidney disease, COVID-19, monitoring algorithm, risk stratification, proteinuria, prognosis.

DEZVOLTAREA UNUI ALGORITM CLINIC DE MONITORIZARE ȘI DE PROGNOSTIC LA PACIENȚII CU BOALĂ CRONICĂ DE RINICHI ÎN PERIOADA POST-COVID

Introducere Boala cronică de rinichi și infecția COVID-19 reprezintă o situație critică prin suprapunerea factorilor de risc major, cu toate acestea monitorizarea renală post-COVID-19 rămâne insuficient standardizată. În acest context, a fost elaborat și validat intern un algoritm clinic de stratificare a riscului, destinat monitorizării și evaluării prognostice a pacienților cu boală cronică de rinichi în perioada post-COVID-19.

Materiale și metode Studiu prospectiv de cohortă care a inclus 280 de pacienți cu boală cronică de rinichi în stadiile G2-G5, nedializați: 140 pacienți post-COVID-19 și 140 martori. La 6-12 luni s-au evaluat rata estimată de filtrare glomerulară, proteinuria și markerii inflamatori, metabolici și hematologici. Au fost înregistrate severitatea COVID-19 și apariția leziunii renale acute în faza activă a infecției. Predictorii asociați stadiilor avansate de boală cronică de rinichi (G4-G5) au fost identificați prin regresie logistică multivariată; performanța algoritmului a fost evaluată prin calibrare și analiza curbei ROC.

Rezultate Declinul funcției renale a fost comparabil între grupuri. La pacienții post-COVID-19, proteinuria persistentă, nivelul crescut al fosfatului seric și hemoglobina scăzută au reprezentat predictori independenți pentru stadiile avansate de boală cronică de rinichi (OR 5,0; 1,8; 1,4; $p<0,005$). Modelul a demonstrat o bună validitate internă (aria sub curbă de aproximativ 0,88).

Concluzii Infecția cu COVID-19 acționează predominant ca un factor de amplificare a vulnerabilității renale. Un algoritm structurat, bazat pe stratificarea riscului, poate îmbunătăți detectarea precoce a deteriorării și poate susține managementul personalizat.

Cuvinte-cheie Boală cronică de rinichi, COVID-19, algoritm de monitorizare, stratificarea riscului, proteinurie, prognostic.

INTRODUCTION

COVID-19 is a systemic inflammatory disease with frequent renal involvement. High ACE2 expression in renal tissue explains the high incidence of COVID-19-associated acute kidney injury (AKI), reported in up to 30% of hospitalized patients and over 50% of those requiring intensive care. COVID-associated AKI results from combined viral, inflammatory, thrombotic, and hemodynamic mechanisms and is associated with adverse outcomes (1, 2).

Chronic kidney disease (CKD) constitutes a major global public health burden and was a key vulnerability factor during the COVID-19 pandemic (3). CKD patients experienced higher rates of severe infection, hospitalization, and mortality. Moreover, SARS-CoV-2 infection may precipitate incomplete AKI recovery, trigger de novo CKD, or accelerate progression of pre-existing CKD (4,5). Longitudinal studies demonstrate persistent eGFR decline, albuminuria, and increased major adverse kidney events among COVID-19 survivors, particularly following severe disease or ICU admission (4,5).

However, consensus-based and evidence-driven protocols specifying follow-up intervals, risk stratification criteria, and core renal biomarkers after coronavirus disease 2019 remain insufficiently defined (5,6). Current recommendations are heterogeneous, and a substantial proportion of patients with COVID-associated AKI remain without nephrologist follow-up, which delays detection of CKD progression.

Objective: To identify independent predictors of advanced chronic kidney disease after coronavirus disease 2019, to evaluate their prognostic performance using multivariate modeling, and to construct and internally validate a risk-stratified clinical monitoring algorithm based on these predictors.

MATERIALS AND METHODS

Study design and population

We conducted a prospective, analytical observational cohort study including 280 adult patients with CKD, enrolled from nephrology clinics between 2021 and 2022." Patients were aged 18–70 years and were not receiving dialysis and had not undergone kidney transplantation and were divided into two equal groups: 140 CKD patients recovered from confirmed COVID-19 (≥30 days post-recovery) and 140 CKD patients without a history of COVID-19. ***Inclusion criteria*** were: age 18-70 years; established CKD of any etiology defined by eGFR <60 mL/min/1.73 m^2 (CKD stages G3a-G5) and/or persistent proteinuria for >3 months; and, for the post-COVID group, documented SARS-CoV-2 infection confirmed by PCR or antigen testing, or based on clinical diagnosis followed by documented recovery. ***Exclusion criteria*** included end-stage renal disease requiring renal replacement therapy, kidney transplantation, active malignancy or active tuberculosis, and inability to provide informed consent or to complete the follow-up period.

Ethical approval

The study protocol was reviewed and approved by the Research Ethics Committee of "Nicolae Testemițanu" State University of Medicine and Pharmacy, Chișinău, Republic of Moldova (Approval No. 6, 18 June 2023). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and applicable national regulations. Written informed consent was obtained from all participants prior to enrollment.

Clinical and laboratory assessment

Demographic data and comorbidities were recorded. In patients after COVID-19, acute infection characteristics were extracted from medical records, including

disease severity (mild, moderate, severe), AKI occurrence (KDIGO criteria), and dialysis requirement. Evaluations were performed 6-12 months after COVID-19 infection (median 9 months) or at a comparable time point in controls. Assessments included renal function (serum creatinine, eGFR by CKD-EPI; cystatin C measured in 60% of patients), urine protein-to-creatinine ratio (persistent proteinuria defined as ≥0.3 g/g), inflammatory markers (CRP, ESR, ferritin), hematologic parameters (hemoglobin), metabolic and mineral markers (albumin, calcium, phosphate, iPTH), and electrolytes.

Cardiac status was systematically evaluated using the New York Heart Association functional classification based on structured clinical interview and resting electrocardiographic assessment. Transthoracic echocardiography was performed according to predefined criteria, including symptoms suggestive of heart failure, abnormal electrocardiographic findings, known structural heart disease, or elevated natriuretic peptides. Quality of life was assessed in all participants using the validated Short Form-36 questionnaire administered by trained personnel at the follow-up visit. All analyses were performed in accredited laboratories using standardized methods, and both groups received standard CKD care.

Statistical analysis and algorithm development

Comparisons between groups were performed using Student's *t*-test or Mann–Whitney *U* test for continuous variables and chi-square test for categorical variables. Adverse kidney prognosis was defined as CKD stage G4-G5 (eGFR <30 mL/min/1.73 m^2) and/or >25% eGFR decline. Independent predictors were identified by multivariable logistic regression with adjusted odds ratios (95% CI). Model calibration and discrimination were assessed using Hosmer-Lemeshow test and ROC-AUC analysis ($p<0.05$). Based on significant predictors, a risk-stratified algoritm for CKD patients after COVID-19 monitoring algorithm was developed and internally validated.

RESULTS

Patient characteristics and comparative outcomes

A total of 280 CKD patients were included: 140 post-COVID (mean age 59.3±10.8 years; 52% male) and 140 non-COVID controls (58.1±11.5 years; 50% male). Baseline demographics, CKD etiologies (chronic glomerulonephritis 32%, diabetic kidney disease 28%, hypertensive nephrosclerosis 22%, polycystic kidney disease 5%, others 13%), and comorbidities (hypertension ~85%, diabetes ~40%, ischemic heart disease ~30%) were comparable between groups. In the post-COVID cohort, disease severity was mild in 20%, moderate in 54%, and severe in 26%; AKI occurred in 27% (5% stage 3), and 7% required acute dialysis. Median time since infection was 9 months (range 1-18 months).

At follow-up, renal function did not differ significantly between post-COVID and control groups (eGFR 37.8±15.6 vs 39.5±14.2 mL/min/1.73 m^2; $p=0.40$), nor did CKD stage distribution or rates of advanced CKD (G4-G5: 55% vs 50%; $p=0.45$). Blood pressure control, glycemic status, and electrolyte levels were similar, indicating no overall acceleration of CKD progression after non-severe COVID-19.

Subgroup analyses demonstrated marked heterogeneity. Post-COVID patients with prior AKI showed a higher prevalence of advanced CKD compared to those without AKI (72% vs 45%; $p<0.01$). Similarly, severe COVID-19 was associated with greater eGFR decline from pre-infection baseline (-5.4 vs -1.8 mL/min), consistent with an accelerated renal trajectory in high-severity cases. In contrast, patients with mild COVID-19 exhibited renal outcomes comparable to those of controls, with predominantly stable kidney function during follow-up.

Laboratory and clinical profile and prognostic factors

Inflammatory markers did not differ significantly between groups (CRP 8.5 vs 7.9 mg/L, p=0.6; ESR 28 vs 26 mm/h, p=0.5). However, post-COVID patients more frequently showed CRP >10 mg/L (24% vs 15%), lower serum albumin (~3 g/dL), and more severe anemia (hemoglobin 10.5 vs 11.2 g/dL, p=0.03). Ferritin levels were higher in post-COVID CKD (380 vs 300 µg/L, p=0.04) and inversely correlated with hemoglobin (r=-0.3, p<0.01), indicating a malnutrition–inflammation profile. Hyperphosphatemia (>4.5 mg/dL) was more prevalent post-COVID (30% vs 20%, p=0.07), while calcium and potassium levels were comparable.

Post-COVID CKD patients more often exhibited advanced heart failure symptoms (NYHA II-III: 40% vs 25%, p=0.02), despite a similar left ventricular ejection fraction. The prevalence of arrhythmias did not differ significantly (14% vs 22%, p=0.1), and hepatic, coagulation, and respiratory parameters were comparable. No late thromboembolic events were observed.

Quality-of-life assessment showed lower Short Form-36 vitality scores in patients after COVID-19 (47 vs 55, p=0.01). After adjustment for chronic kidney disease stage, no significant differences were observed in the physical functioning or emotional role domains between groups. In the post-COVID cohort, univariate analysis identified persistent proteinuria, higher serum phosphate, lower hemoglobin, elevated CRP, and prior AKI as predictors of advanced CKD (G4-G5). Multivariable logistic regression identified three independent predictors (Table 1): persistent proteinuria (OR 5.0, 95% CI 2.0-12.5; p<0.001), serum phosphate (OR 1.8 per +1 mg/dL; p<0.001), and hemoglobin (OR 1.4 per -1 g/dL; p=0.005). Age, sex, diabetes, inflammatory markers, and acute COVID severity were not independently associated. The model demonstrated good calibration (Hosmer–Lemeshow p=0.45) and discrimination (AUC 0.88). Patients with all three risk factors uniformly had advanced CKD, whereas those without them remained largely stable.

Table 1. Independent predictors of advanced CKD (Stage G4-G5) in post-COVID patients (Multivariate Logistic Regression, n=140)

Predictor	Adjusted OR (95% CI)	p-value
Persistent proteinuria (yes vs no)	5.0 (2.0 – 12.5)	<0.001
Serum phosphate (per +1 mg/dL)	1.8 (1.3 – 2.5)	<0.001
Hemoglobin (per –1 g/dL)	1.4 (1.1 – 1.8)	0.005
D-dimer (per +1 µg/mL)	1.2 (0.9 – 1.6)	0.10 (NS)
Age (per +10 years)	1.1 (0.9 – 1.3)	0.18 (NS)
Male sex (vs female)	1.3 (0.5 – 3.1)	0.60 (NS)

Note: OR – odds ratio; CI – confidence interval; NS – not significant. Proteinuria is defined as urine protein-creatinine ratio ≥0.3 g/g.

Development of the monitoring algorithm: Based on study findings, we developed a risk-stratified clinical algorithm for management of CKD after COVID-19. Patients are classified as high risk if they have had severe COVID-19 (ICU admission or major complications) or COVID-associated AKI, and/or present adverse prognostic markers at follow-up (persistent proteinuria, serum phosphate >4.5 mg/dL, hemoglobin <11 g/dL). Patients with mild COVID-19 and no high-risk laboratory abnormalities are classified as Low Risk.

High-Risk patients are scheduled for nephrology follow-up at 1, 3, 6, and 12 months post-recovery, including assessment of serum creatinine and eGFR (± cystatin C), urine protein or albumin and sediment, serum phosphate, potas-

sium, hemoglobin, blood pressure, and glycemic control. Low-Risk patients are monitored at 6 and 12 months, in addition to routine CKD care.

Predefined criteria for clinical deterioration included a decline in estimated glomerular filtration rate ≥10% from baseline, new-onset or doubling of proteinuria, uncontrolled blood pressure despite therapy adjustment, serum phosphate >4.5 mg/dL, or a decrease in hemoglobin ≥1 g/dL. The occurrence of any of these criteria prompted nephrology reassessment according to the study protocol. In the high-risk group, at least one deterioration criterion was documented in 30% of patients during follow-up, most commonly a ≥10% decline in estimated glomerular filtration rate within 3 months or doubling of proteinuria. The algorithm prioritizes early nephroprotective interventions, including optimization of RAAS blockade, initiation of SGLT2 inhibitors when indicated, strict glycemic and blood pressure control, dietary sodium and phosphate restriction, and targeted management of anemia and CKD-mineral bone disorder (e.g., phosphate binders, iron supplementation, erythropoiesis-stimulating agents).

To ensure a multidisciplinary approach, High-risk patients are advised to undergo cardiology evaluation at 3-6 months after COVID-19, with ECG and echocardiography as indicated, due to increased heart failure burden. Patients with persistent fatigue or functional impairment are referred for rehabilitation programs to improve physical capacity and quality of life.

The clinical algorithm is summarized in (Table 2). This structure was retrospectively applied to our cohort of patients with CKD after COVID-19: it correctly identified the 35 high-risk patients who experienced significant renal function decline and would have led them to closer surveillance or early intervention, potentially preventing delay in care. Meanwhile, it would have spared low-risk patients from unnecessary frequent visits. No low-risk patient in our study had an adverse event that would have been missed under the algorithm's follow-up scheme, supporting its safety and efficiency.

Table 2. Post-COVID Monitoring Algorithm for CKD Patients

Risk Category	Criteria	Monitoring Plan	Recommended Interventions
High Risk CKD post-COVID (vulnerable to progression)	• Severe COVID-19 (hospitalized, ICU or COVID-AKI) • Adverse renal markers at follow-up: persistent proteinuria, hyperphosphatemia, and/or anemia	Nephrology follow-up at 1, 3, 6, 12 months in first year post-recovery. Each visit: Check serum creatinine (eGFR) ± cystatin C, urinalysis (proteinuria, hematuria), serum phosphate, potassium, hemoglobin, BP, etc.	If any decline in eGFR or abnormal finding, escalate care promptly: • Optimize BP (<130/80) with RAAS blockade (ACEi/ARB) unless contraindicated. • Start SGLT2 inhibitor (if diabetic CKD or eligible) for renal protection. • Treat proteinuria (maximize ACEi/ARB, add finerenone if indicated). • Manage phosphate (dietary restriction, phosphate binders) and CKD-MBD per guidelines. • Correct anemia (iron, EPO stimulating agents if Hb <10). • Ensure strict glycemic control in diabetes (A1c ~7%). • Cardiology consultation at 3–6 months: ECHO & ECG to evaluate post-COVID cardiac function (especially if heart failure history or symptoms). • Rehabilitation for persistent fatigue: graded exercise program, nutritional counseling, and psychological support as needed.
Low Risk CKD post-COVID (likely stable course)	• Mild COVID-19 (outpatient or brief illness) AND • No significant proteinuria, normal phosphate, no severe anemia at follow-up	Follow-up at ~6 months post-COVID (and again at 12 months if stable). Evaluate the same parameters as above.	Continue routine CKD care with primary care/nephrology as per standard schedule. If new abnormalities emerge (e.g., develops proteinuria or eGFR drop), reclassify as High Risk and intensify monitoring & therapy accordingly.

Note: BP – blood pressure; RAAS – renin-angiotensin-aldosterone system; ACEi – angiotensin-converting enzyme inhibitor; ARB – angiotensin receptor blocker; SGLT2 – sodium-glucose cotransporter-2; CKD-MBD – chronic kidney disease mineral-bone disorder; ECHO – echocardiogram; ECG – electrocardiogram.

DISCUSSION

The present study evaluated renal outcomes in patients with chronic kidney disease after coronavirus disease 2019 and developed an internally validated monitoring algorithm based on identified prognostic factors. The main findings are: (1) renal progression after COVID-19 was heterogeneous; (2) severe infection and acute kidney injury were associated with a greater decline in renal function; and (3) persistent proteinuria, elevated serum phosphate, and low hemoglobin independently predicted advanced chronic kidney disease.

Renal outcomes differed according to infection severity. Patients with mild coronavirus disease 2019 showed kidney function trajectories comparable to controls, consistent with previous longitudinal analyses reporting preserved renal function in most survivors (6, 7). In contrast, individuals with severe disease or COVID-19–associated acute kidney injury experienced a greater estimated glomerular filtration rate decline and higher prevalence of advanced chronic kidney disease. This gradient is in agreement with large cohort studies demonstrating increasing risk of chronic kidney disease progression and end-stage kidney disease proportional to the severity of acute infection (8,9).

A subgroup of post-COVID-19 patients exhibited higher ferritin levels, lower albumin, and more pronounced anemia. These findings suggest persistent inflammatory activity, which is a recognized contributor to chronic kidney disease progression. Similar inflammatory patterns have been described in post-acute coronavirus disease 2019 syndromes (15, 16). However, in the multivariable analysis, inflammatory markers were not independent predictors, indicating that their prognostic impact is likely mediated through renal functional parameters.

Multivariate modeling identified three independent predictors of advanced chronic kidney disease: persistent proteinuria, serum phosphate concentration, and hemoglobin level. These factors are well-established determinants of chronic kidney disease progression irrespective of infection status. Persistent proteinuria reflects ongoing glomerular injury, hyperphosphatemia contributes to vascular and renal structural damage, and anemia promotes tissue hypoxia and fibrosis. The lack of independent associations with age, diabetes, or acute infection severity suggests that current biological status is more relevant than prior infection characteristics in determining medium-term renal prognosis.

Based on these findings, we developed a risk-stratified monitoring algorithm integrating infection severity and laboratory predictors. This strategy is consistent with the Kidney Disease: Improving Global Outcomes recommendations for post–acute kidney injury surveillance (10-12), while extending risk stratification by incorporating proteinuria, phosphate, and hemoglobin. Early identification of high-risk patients may facilitate timely nephrology follow-up and optimization of nephroprotective therapy, which is known to delay progression and reduce mortality (13, 14).

Our results align with population-level analyses showing increased adverse kidney events after coronavirus disease 2019 (17, 18). Unlike machine-learning prediction models requiring complex inputs (19–21), the proposed algorithm relies on routinely available clinical parameters, which enhances feasibility in daily practice.

Overall, progression of chronic kidney disease after coronavirus disease 2019 was not universal but concentrated in identifiable high-risk subgroups. Structured monitoring based on modifiable renal risk factors may support more efficient allocation of healthcare resources.

Limitations

Several limitations should be acknowledged. Follow-up duration was limited (median approximately 9 months), and longer observation is required to determine long-term renal trajectories. The observational design limits causal inference. External validation of the algorithm is necessary, as current performance metrics are based on internal validation. Classification of infection severity relied on medical documentation, which may introduce misclassification bias. Finally, emerging biomarkers and genetic factors were not evaluated.

CONCLUSIONS

1. In patients with chronic kidney disease, renal outcomes after coronavirus disease 2019 were heterogeneous. Mild infection was not associated with significant changes in kidney function compared with controls, whereas severe infection and coronavirus disease 2019-associated acute kidney injury were associated with a greater decline in estimated glomerular filtration rate and higher prevalence of advanced chronic kidney disease.
2. Persistent proteinuria, elevated serum phosphate, and low hemoglobin were independent predictors of advanced chronic kidney disease after COVID-19. These parameters may be used to identify patients at increased risk of progression during follow-up.
3. A structured monitoring strategy based on infection severity and these laboratory predictors may facilitate earlier nephrology reassessment and optimization of nephroprotective therapy, while allowing stable patients to be followed at standard intervals.

CONFLICT OF INTEREST

The authors declare no conflicts of interest related to this study. No financial or personal relationships influenced the conduct or outcomes of this research.

ETHICAL APPROVAL

The research protocol was approved by the Institutional Ethics Committee of “Nicolae Testemițanu” State University of Medicine and Pharmacy (Approval No. 6 of 18/06/2023). All participants provided informed consent prior to enrollment. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and Good Clinical Practice guidelines. Participant confidentiality was strictly maintained throughout the study.

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